REMARKS

Claims 21, 25-28, 30 and 32-35 are pending and allowed. New claims 36-41 have been added. Accordingly, following entry of the amendments made herein, claims 21, 25-28, 30, and 32-41 will be pending in the present application.

The Examiner has requested a copy of the Amendment filed in connection with the above-identified application during the pendency of Interference No. 105,168. Accordingly, the Amendment is submitted herewith as Exhibit A attached hereto.

New claims 36-41 were previously submitted in connection with the above-mentioned Amendment. A motion (Kroczek Contingent Motion 3) requesting entry of the Amendment was granted by the Board of Patent Appeals and Interferences (see page 8 of the Decision on Interlocutory Motions submitted herewith as Exhibit B).

Support for the new claims may be found in the present application at, for example, on page 7, lines 25-46, in claim 1 as filed, on page 12, line 20, on page 13, line 1, and in the Statement Of Applicant Regarding Permanence And Availability Of Deposited Microorganisms, originally submitted on October 15, 2002 in connection with related application no. 09/509,283.

Claims 21 and 30 have been amended. Support for the recitation in amended claims 21 and 30 that the 8F4 polypeptide occurs on two-signal-activated human "$CD4^+$ T lymphocytes from human peripheral blood" can be found, *e.g.*, on page 12, line 20 and page 12, line 1 of the specification. Claims 21 and 30 have also been amended to clarify that the 8F4 polypeptide is recognized by the antibody "produced by the hybridoma" deposited with the DSMZ and assigned accession no. DSM ACC2539, since the hybridoma, not the antibody itself, was the subject matter of the DSMZ deposit (see Statement of Applicant Regarding Permanence and Availability of Deposited Microorganisms submitted in connection with related application no. 09/509,283 on October 15, 2002).

No new matter is added.

CONCLUSION

Applicant respectfully requests that the amendment and remarks made herein be entered and made of record in the file history of the subject application.

Respectfully submitted,

Date: November 23, 2005

Nikolaos C. George 39,201

Nikolaos C. George (Reg. No.)
JONES DAY
222 E. 41st Street
New York, New York 10017
(212) 326-3939

by: Nina Abrishamkar
Limited Recognition
L0012

APPENDIX B

AMENDMENT TO BE ENTERED

IF KROCZEK CONTINGENT MOTION 3 IS GRANTED

(A

Express Mail No.: EV 531 695 995 US

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application of: Kroczek, Richard

Confirmation No.: 8614

Application No.: 09/823,307

Art Unit: 1644

Filed: April 2, 2002

Examiner: Roark, Jessica H.

For: METHODS OF MODULATING T LYMPHOCYTE COSTIMULATION

Attorney Docket No.: 7853-235

AMENDMENT

Mail Stop INTERFERENCE
Board of Patent Appeals and Interferences
U.S. Patent and Trademark Office
PO Box 1450
Alexandria, Virginia 22313-1450

Sir:

This Amendment is being filed in during the pendency of Patent Interference No. 105,168. Applicant respectfully requests entry of this Amendment and consideration of the Remarks made herein in connection with the above-captioned application. Applicant submits herewith an Amendment Fee Transmittal Sheet as Appendix C.

AMENDMENTS TO THE CLAIMS are reflected in the listing of the claims which begins on page 2 of this paper.

REMARKS begin on page 7 of this paper.

AMENDMENTS TO THE CLAIMS

This listing of claims will replace all prior versions, and listings, of claims in the application:

LISTING OF CLAIMS

1-20. (Canceled)

21. (Previously presented) A method of inhibiting costimulation of human T lymphocytes comprising: contacting a human T lymphocyte with a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated human T lymphocytes;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539,

such that costimulation of the human T lymphocyte is inhibited.

22-24. (Canceled)

25. (Previously presented) The method of claim 21, wherein the monoclonal antibody recognizes the human 8F4 polypeptide of about 55 kilodaltons to 60 kilodaltons, as determined by non-reducing SDS-PAGE.

26. (Previously presented) The method of claim 21, wherein the monoclonal antibody recognizes the peptide chain of about 27 kilodaltons, as determined by reducing SDS-PAGE.

27. (Previously presented) The method of claim 21, wherein the monoclonal antibody recognizes the peptide chain of about 29 kilodaltons, as determined by reducing SDS-PAGE.

28. (Previously presented) The method of claim 21, wherein the monoclonal antibody recognizes a human 8F4 polypeptide present on human $CD4^+$ T lymphocytes and activated human $CD8^+$ T lymphocytes.

29. (Canceled)

30. (Previously presented) A method of inhibiting rejection of an organ transplant, comprising: administering to an individual in need of such inhibition an 8F4 inhibitory molecule, which 8F4 inhibitory molecule is a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated human T lymphocytes;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody deposited with the DSMZ and assigned accession no. DSM ACC2539,

in an amount sufficient to inhibit rejection of an organ transplant.

31. (Canceled)

32. (Previously presented) The method of claim 30, wherein the monoclonal antibody recognizes the human 8F4 polypeptide of about 55 kilodaltons to 60 kilodaltons, as determined by non-reducing SDS-PAGE.

33. (Previously presented) The method of claim 30, wherein the monoclonal antibody recognizes the peptide chain of about 27 kilodaltons, as determined by reducing SDS-PAGE.

34. (Previously presented) The method of claim 30, wherein the monoclonal antibody recognizes the peptide chain of about 29 kilodaltons, as determined by reducing SDS-PAGE.

35. (Previously presented) The method of claim 30, wherein the monoclonal antibody recognizes a human 8F4 polypeptide present on activated human $CD4^+$ T lymphocytes and activated human $CD8^+$ T lymphocytes.

36. (New) A method for treating an immune disorder, comprising: administering to an individual in need of treatment a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated $CD4^+$ T lymphocytes from human peripheral blood;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody produced by the hybridoma deposited with the DSMZ and assigned accession no. DSM ACC2539,

in an amount sufficient to ameliorate a symptom of the immune disorder, such that the immune disorder is treated.

37. (New) A method for treating an autoimmune disorder, comprising: administering to an individual in need of treatment a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated $CD4^+$ T lymphocytes from human peripheral blood;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody produced by the hybridoma deposited with the DSMZ and assigned accession no. DSM ACC2539,

in an amount sufficient to ameliorate a symptom of the autoimmune disorder, such that the autoimmune disorder is treated.

39. (New) A method for treating cancer, comprising: administering to an individual in need of treatment a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated $CD4^+$ T lymphocytes from human peripheral blood;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody produced by the hybridoma deposited with the DSMZ and assigned accession no. DSM ACC2539,

in an amount sufficient to ameliorate a symptom of the cancer, such that the cancer is treated.

40. (New) A method for treating a chronic viral disease, comprising: administering to an individual in need of treatment a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated $CD4^+$ T lymphocytes from human peripheral blood;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody produced by the hybridoma deposited with the DSMZ and assigned accession no. DSM ACC2539,

in an amount sufficient to ameliorate a symptom of the chronic viral disease, such that the chronic viral disease is treated.

41. (New) A method for treating AIDS, comprising: administering to an individual in need of treatment a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:

a) is an inducible T cell costimulatory molecule;

b) occurs on two-signal-activated $CD4^+$ T lymphocytes from human peripheral blood;

c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);

d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

e) is recognized by the antibody produced by the hybridoma deposited with the DSMZ and assigned accession no. DSM ACC2539,

in an amount sufficient to ameliorate a symptom of AIDS, such that AIDS is treated.

REMARKS

Claims 21, 25-28, 30 and 32-35 are pending and allowed subject to interference in this application. New claims 36-41 have been added. Accordingly, following entry of the amendments made herein, claims 21, 25-28, 30, 32-41 will be pending in the present application.

Support for the new claims may be found in the present application at, for example, on page 7, lines 25-46, in claim 1 as filed, on page 12, line 20, on page 13, line 1, and in the Statement Of Applicant Regarding Permanence And Availability Of Deposited Microorganisms, originally submitted on October 15, 2002 in connection with Kroczek Application No. 09/509,283 and attached hereto as Appendix D.

No new matter is added.

CONCLUSION

Applicant respectfully requests that the amendments and remarks made herein be entered and made of record in the file history of the subject application.

Respectfully submitted,

Date

April 12, 2005

Nikolaos C. George 39,201

Nikolaos C. George (Reg. No.)

JONES DAY
222 E. 41st Street
New York, New York 10017
(212) 326-3939

by: Muna Abu Sharr
Limited Recognition
Under 37 CFR § 11.9(b)
Copy of Certificate Enclosed

BEFORE THE OFFICE OF ENROLLMENT AND DISCIPLINE
UNITED STATES PATENT AND TRADEMARK OFFICE

LIMITED RECOGNITION UNDER 37 CFR § 11.9(b)

Dr. Abu-Shaar is hereby given limited recognition under 37 CFR §11.9(b) as an employee of Jones Day to prepare and prosecute patent applications wherein the patent applicant is the client of Jones Day, and the attorney or agent of record in the applications is a registered practitioner who is a member of Jones Day. This limited recognition shall expire on the date appearing below, or when whichever of the following events first occurs prior to the date appearing below: (i) Dr. Abu-Shaar ceases to lawfully reside in the United States, (ii) Dr. Abu-Shaar's employment with Jones Day ceases or is terminated, or (iii) Dr. Abu-Shaar ceases to remain or reside in the United States on an H-1B visa.

This document constitutes proof of such recognition. The original of this document is on file in the Office of Enrollment and Discipline of the U.S. Patent and Trademark Office.

Expires: September 30, 2005

Harry I. Moatz
Director of Enrollment and Discipline

Paper 139

Filed by: Interference Trial Section Motions Panel
Mail Stop Interference
P.O. Box 1450
Alexandria Va 22313-1450
Tel: 571-272-9797
Fax: 571-273-0042

Entered: May 23, 2005

UNITED STATES PATENT AND TRADEMARK OFFICE

BEFORE THE BOARD OF PATENT APPEALS AND INTERFERENCES

RICHARD KROCZEK
Junior Party
(Applications 09/509,283, 09/823,307, and 09/972,524),

v.

TAKUYA TAMATANI and KATSUNARI TEZUKA
Senior Party
(Applications 09/383,551, 09/561,308, and 10/301,056).

Patent Interference No. 105,168

Before: SCHAFER, LEE and MOORE, Administrative Patent Judges,[1]
SCHAFER, Administrative Patent Judge.

Decision - Interlocutory Motions - Bd.R. 125(b)

The following preliminary motions are before us: (1) the parties Joint Motion 2 to redefine the interfering subject matter (Paper 129); (2) Contingent Joint Motion 3 to redefine the interfering subject matter (Paper 131); (3) Tamatani unopposed contingent Motion 3 to add claims to Tamatani's involved application 09/561,308 (Paper 133); and (4) Kroczek unopposed contingent Motion 3 to add claims to Kroczek involved application 09/823,307 (Paper 136).

[1] As part of Board efforts under the Government Paperwork Elimination Act, signatures on papers originating from the Board are being phased out in favor of a completely electronic record. Consequently, subsequent papers in this case originating at the Board will not have signatures. The signature requirements for the parties have not changed. See, e.g., 37 C.F.R. § 10.18.

Joint Motion 2 is denied. We grant contingent Joint Motion 3, Tamatani Contingent Motion 3 and Kroczek Contingent Motion 3.

Joint Motion 2

The parties seek to substitute three counts for the current count. The current count (Count 1) is:

> The method according to Claim 21 of Kroczek application 09/823,307
> or
> the method according to Claim 91 of Tamatani application 09/383,551.

Claim 21 of the Kroczek 307 application states:

> 21. A method of inhibiting costimulation of human T lymphocytes comprising: contacting a human T lymphocyte with a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:
>
> a) is an inducible T cell costimulatory molecule;
> b) occurs on two-signal-activated human T lymphocytes;
> c) exhibits a molecular weight of about 55 to 60 kilodaltons as determined by non-reducing sodium dodecyl sulphate polyacrylamide gel electrophoresis (SDS-PAGE);
> d) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and
> e) is recognized by the antibody deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539,
>
> such that costimulation of the human T lymphocyte is inhibited.

Claim 91 of the Tamatani 551 application, rewritten in independent form, provides:

> 91. A method of inhibiting activation of lymphocytes in a subject, the method comprising administering to the subject an effective amount of the pharmaceutical composition comprising (1) a purified chimeric, humanized or human monoclonal antibody or a portion thereof that binds to the extracellular region to a polypeptide consisting of SEQ ID NO:2. and (2) a pharmaceutically acceptable carrier.

The parties' involved claims, i.e. those which have been designated as corresponding to the count, relate to a polypeptide called human inducible costimulatory molecule (ICOS),[2] an antibody

---

[2] The parties specification have each referred to the human ICOS molecule by a different name. Kroczek calls it human 8F4 polypeptide. Tamatani calls it human JTT-1 polypeptide.

which binds to ICOS, pharmaceutical compositions which include the antibody, methods of treatment using the antibody, methods of producing the antibody and hybridomas or cells which produce the antibody.

The parties jointly urge that their claims include three separately patentable inventions: (1) the method of treatment using the ICOS antibody, (2) the ICOS antibody and (3) ICOS or the nucleic acid which expresses ICOS. These inventions are reflected in three proposed counts:

Proposed Count 1[3]

> A method of inhibiting costimulation of human T lymphocytes comprising: contacting a human T lymphocyte with a monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:
> (a) occurs on two-signal-activated CD4+ T lymphocytes from human peripheral blood;
> (b) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and
> (c) is recognized by the antibody produced by the hybridoma deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539, such that costimulation of the human T lymphocyte is inhibited.
>
> OR
>
> A method of inhibiting activation of lymphocytes in a subject, the method comprising administering to the subject an effective amount of the pharmaceutical composition comprising (1) a purified chimeric, humanized or human monoclonal antibody or a portion thereof that binds to the extracellular region to a polypeptide consisting of SEQ ID NO:2 [of Tamatani Application 09/383,551] and (2) a pharmaceutically acceptable carrier.

Proposed Count 2

> A murine monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:
> (a) occurs on two-signal-activated CD4+ T lymphocytes from human peripheral blood;

---

[3] The second alternative of the count, as proposed by the parties, referenced a number of Tamatani claims and was essentially the subject matter of Claim 91 of Tamatani Application 09/383,551. That claim is a dependent claim which incorporates by reference the subject matter of a number of parent claims. For clarity it has been rewritten in independent form with the express reference to the 551 application added to clearly identify the source of SEQ ID NO:2.

(b) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

(c) is recognized by the antibody produced by the hybridoma deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539.

OR

A purified non-hamster antibody or portion thereof that binds to a polypeptide consisting of SEQ ID NO: 2 [of Tamatani Application 09/383,551].

Proposed Count 3

An isolated human 8F4 polypeptide, wherein said 8F4 polypeptide:

(a) occurs on two-signal-activated CD4+ T lymphocytes from human peripheral blood;

(b) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

(c) is recognized by the antibody produced by the hybridoma deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539;

OR

An isolated polypeptide comprising the amino acid sequence set forth in SEQ ID NO: 2.

OR

An isolated nucleic acid comprising a nucleotide sequence that encodes a polypeptide comprising the amino acid sequence of SEQ ID NO: 2 [of Tamatani Application 09/383,551].

Discussion

Where an interference includes multiple counts each count must represent a separately patentable invention. 37 CFR § 41.201. Thus, when a party seeks to have the interference proceed with additional counts, each count must be shown to be patentably distinct from each of the other counts. In making this analysis, each proposed count is presumed, in turn, to be prior art with respect to the others. The remaining counts are evaluated in light of the presumed prior art for anticipation and obviousness. Both determinations are made from the perspective of the person of ordinary skill in the art.

With respect to proposed Counts 2 and 3 (directed to the antibody and the polypeptide, respectively), the parties allege that the recitation of an antibody to human ICOS polypeptide does not disclose the human ICOS polypeptide itself or the nucleic acid encoding that polypeptide.

Comparing the express "teachings" of proposed Counts 2 and 3, it appears that the first alternative of each proposed count fully describes both the antibody and the polypeptide. Both counts describe the antibody as produced by the same deposited hybridoma assigned Accession No. DSM ACC2539. Both proposed Counts 2 and 3 also fully describe the same characteristics of the 8F4 polypeptide. Thus, each first alternative of proposed Counts 2 and 3 anticipates the other. Proposed Counts 2 and 3 have not been shown to be patentably distinct. Joint Motion 3 is denied.

Joint Contingent Motion 3

The parties have also filed a joint contingent motion with two proposed counts. One is directed to the method of treatment that appears to be identical to proposed Count 1 discussed above. The second count is essentially a combination of proposed Counts 2 and 3 above. It includes alternatives to the antibody, the polypeptide and the nucleic acid:

> A murine monoclonal antibody that recognizes a human 8F4 polypeptide, wherein said 8F4 polypeptide:
>
> (a) occurs on two-signal-activated CD4+ T lymphocytes from human peripheral blood;
>
> (b) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and
>
> (c) is recognized by the antibody produced by the hybridoma deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539.
>
> OR
>
> A purified non-hamster antibody or portion thereof that binds to a polypeptide consisting of SEQ ID NO: 2 [of Tamatani Application 09/383,551].
>
> OR
>
> An isolated human 8F4 polypeptide, wherein said 8F4 polypeptide:
>
> (a) occurs on two-signal-activated CD4+ T lymphocytes from human peripheral blood;
>
> (b) is a dimer of two peptide chains exhibiting molecular weights of about 27 kilodaltons and 29 kilodaltons, as measured by reducing SDS-PAGE; and

(c) is recognized by the antibody produced by the hybridoma deposited with the Deutsche Sammlung von Mikroorganismen und Zellkulturen GmbH ("DSMZ") and assigned accession no. DSM ACC2539;

OR

An isolated polypeptide comprising the amino acid sequence set forth in SEQ ID NO: 2 [of Tamatani Application 09/383,551].

OR

An isolated nucleic acid comprising a nucleotide sequence that encodes a polypeptide comprising the amino acid sequence of SEQ ID NO: 2 [of Tamatani Application 09/383,551].

We will refer to this count as the composition count.

The parties assert that the subject matter of composition count does not anticipate or render obvious the subject matter of the treatment count.

Discussion

Taking the composition count as presumed prior art, it is correctly argued that the method steps specified in the treatment count are not described. Thus, the composition count does not anticipate the treatment count. The steps are also said not to be obvious from the composition count because the inhibition of the activation of lymphocytes by the antibodies of the composition count is unpredictable:

> Not all anti-human ICOS antibodies can inhibit the activation or costimulation of T lymphocytes .... Indeed, an anti-human ICOS antibody has uses other than inhibiting the activation or costimulation of human T lymphocytes .... The mere disclosure of an antibody to human ICOS, as by [the composition count], cannot fairly be construed to suggest any particular method for using the antibody, much less the specific method of inhibiting T lymphocyte activation or costimulation recited in [the treatment count] ... .

Paper 131, pp. 8-9 (citations to proposed statements of facts deleted, bracketed material added). The parties further argue that when the subject matter of the proposed composition count is considered in the context of the relevant prior art there is no suggestion or motivation to use the antibody of composition count in the specific method of the treatment count with a reasonable expectation of success. Paper 131, p. 9.

The parties argue similarly with respect to the alternatives of the composition count specifying the ICOS polypeptide and the nucleic acid which encodes ICOS:

> [T]he human ICOS polypeptide was not known or suggested to be involved in the activation or costimulation of T lymphocytes . . . . Without such knowledge or suggestion, there could not have been any motivation to use the human ICOS polypeptide or the nucleic acid encoding the polypeptide to make and use anti-human ICOS antibodies in a method of inhibiting activation or costimulation of T lymphocytes, and one of ordinary skill in the art would not have reasonably believed that an antibody to human ICOS would inhibit the activation or costimulation of T lymphocytes.

Paper 131, p. 11 (citations to statement of proposed facts omitted).

The parties support their arguments with the declaration testimony of Prof. Jeffrey A. Bluestone, Ph.D. Paper 130. The declaration establishes Prof. Bluestone as an expert in the field of the invention –immunobiology. Paper 130, p. 2, ¶ 3. We find that Prof. Bluestone's testimony relating to the patentable distinctness of the treatment and the composition count to be highly credible.

Prof. Bluestone testifies that the mere disclosure of an anti-human ICOS antibody, as in the composition count, does not suggest any particular method of using the antibody. Paper 130, pp. 3-4, ¶ 8. He further testifies that this was the state of the art prior to September, 1997, as well as on the date of his testimony. Paper 130, pp. 3-4, ¶ 8. Prof. Bluestone also testifies that as of the date of his testimony it was known that only some anti-ICOS antibodies can inhibit the activation and costimulation of T lymphocytes. Paper 130, p. 7, ¶ 16. According to Prof. Bluestone, it was also known that some antibodies to costimulatory proteins other than ICOS inhibit T lymphocyte costimulation while others did not. Paper 130, pp. 6-7, ¶ 15. Prof. Bluestone further testifies that knowledge of the structure of ICOS alone, would not have suggested that ICOS is involved in human T cell costimulation. Paper 130, p. 4, ¶ 10. He also discusses some highly relevant prior art relating to the structure of ICOS and testifies that even with this additional knowledge and the general knowledge in the art, one of ordinary skill in the art would not conclude "that human ICOS polypeptide was involved in the activation or costimulation of T lymphocytes. Paper 130, pp. 4-6, ¶¶ 11-14.

The testimony establishes that the inhibition or activation or costimulation of T lymphocytes is unpredictable. Therefore, there would be no reasonable expectation of success that the human anti-ICOS antibody of the composition count would inhibit the activation or costimulation of T lymphocytes. Thus, the subject matter of the composition count would not have suggested that ICOS

antibodies could affect the costimulation of T lymphocytes. We conclude, therefore, that the subject matter of the treatment count would not be obvious from the subject matter of composition count. The parties contingent Joint Motion 3 is granted. The interference will be redeclared in a separate paper.

Tamatani Contingent Motion 3 and Kroczeck Contingent Motion 3

Each of the parties has filed a motion contingent on the grant of the parties joint contingent Motion 3. Kroczek has filed a motion to add Claims 36-41 to involved Kroczek Application 09/823,307. Paper 136. Tamatani has filed a motion to add Claims 131-136 to Tamatani involved Application 09/561,308. Paper 133. Both motions are unopposed.

We grant these motions to the extent that the amendments will be entered into the respective application files. In granting the motions we express no opinion on the patentability of the newly added claims (including compliance with 35 U.S.C. § 112). Thus, the granting of the motions is without prejudice to further examination by a primary examiner when the applications are returned to the jurisdiction of the examining corps.

ORDER

It is

ORDERED that the parties joint motion to substitute three counts for the current count is DENIED;

FURTHER ORDERED that the parties joint motion to substitute two counts for the current count is GRANTED;

FURTHER ORDERED that the parties' unopposed motions to file an amendment to add claims to applications 09/823,307 and 09/561,308 is GRANTED;

FURTHER ORDERED that this interference be redeclared consistent with this decision; and

FURTHER ORDERED that a copy of this decision and opinion be made of record in each of the involved applications.

cc (FAX):

Attorneys for Kroczek:
Samuel B. Abrams
JONES DAY
222 East 41st Street
New York, NY 10017
Tel: 212-326-3939
Fax: 212-755-7306

Attorneys for Tamatani:
John Kilyk, Jr.
LEYDIG, VOIT & MAYER, LTD.
Two Prudential Plaza, Suite 4900
180 N. Stetson Avenue
Chicago, IL 60601
Tel: 312-616-5600
Fax: 312-616-5700